The Newsletter of the Australian
and New Zealand Society of
Paediatric Dentistry

Synopses

OCTOBER 2005, ISSUE 32

www.anzspd.org.au

A pain in the pulp: innervation, inflammation and management of the compromised primary tooth pulp

Summary of a lecture given to the Australian Academy of Paediatric Dentistry, 3rd March 2005, Adelaide

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Introduction

The tooth pulp provides us with a unique opportunity to study mechanisms of peripheral innervation, inflammation and pain. Animal research allows us to study both peripheral and central changes following pulpal injury, whereas the use of human tissue enables us to correlate histological findings with a known pain history.

One of the single most important factors to further our knowledge of tooth innervation has been the development of sophisticated immunohistochemical staining techniques. These have enabled us to identify individual nerve subpopulations according to their expression of a host of different neurotransmitters, ion channels and receptors. Notably, nerves can be classified according to their expression of specific neuropeptides. These are biologically active proteins with a wide range of regulatory functions including: pain perception, vasoregulation, immune cell regulation, and bone metabolism. At least 16 different peptidergic (neuropeptide- containing) fibres have been identified in tooth pulp since the 1970s, and more are being discovered all the time. One of the most interesting neuropeptides is substance P, because of its key role in pain processing, vasodilation and neurogenic inflammation. Study of these neuropeptide-expressing nerves is giving us a much greater insight, not only into the diverse anatomy of dental innervation, but also the function of pulpal nerves.

The tooth pulp is one of the most densely innervated human tissues and predominantly comprises sensory (nociceptive) fibres. Traditionally, we have thought that intradental nerves served the sole purpose of pain perception but we are now appreciating that their primary function may actually relate to immunoregulation and healing.

Primary tooth pulp innervation and inflammation

A question frequently asked, in relation to tooth pulp innervation, is whether primary teeth are less sensitive than permanent teeth? Anecdotally, many clinicians have felt this to be the case. However, publications on primary tooth innervation and sensibility are sparse. These studies have suggested that primary tooth pulp innervation is less dense than that seen in permanent teeth, and may comprise fewer myelinated fibres.

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THIS ISSUE

President's Report

It is an interesting dichotomy, dental care and medical care.

The political and economic factors that forced the disembodied oral cavity and dental care onto the individual States, while leaving the rest of the body and medicine with the Federal Government probably saved the community and the profession from a calamity like the UK NHS Dental Scheme. At the time the Whitlam Government introduced the original Medibank more than thirty years ago, the distinction between dental care and medical care was sharply defined. Oral disease was highly prevalent, chronic, progressively degenerative, expensive and unpredictable to treat. Medical conditions on the other hand were less prevalent, mostly acute, and affected individuals would (conveniently) either get better or die!

While differences clearly remain, the distinctions between medical and dental care at a public health level, have diminished. Oral disease is markedly less prevalent and more predictable though still expensive to treat. The medical paradigm of 'heal-or-die' has irrevocably changed, causing the cost of universal health care to blow out, as successful medical treatments have raised expectations for the management of chronic disease and disability. A thought-provoking statistic is that roughly half of total health care expenditure is spent during the last year of a patient's life.

Every dental student studies the basics of medicine and surgery to learn to recognise the oral manifestations of systemic disease, the systemic ramifications of oral disease, and the ways in which medical conditions and treatment can cause or modulate oral disease, affect oro-facial growth and development, and complicate our therapeutic interventions. The oral cavity cannot be managed in isolation from the rest of the body.

So why is it that for a preschool aged child, a boil on the bum is regarded so differently to a boil on the gum? Because the former is regarded as a 'valid' medical problem, surgical drainage would be established under the aegis of our universal medical cover, including the cost of hospital admission and a general anaesthetic if required. Parents could elect to have private treatment, but would still receive their Medicare rebate.

A dental abscess however, falls under an entirely different jurisdiction. There is no automatic universal access to the dental treatment that would resolve this common problem, and even less access to timely interventions that could have avoided an abscess in the first place. If an anaesthetic were required, fully private treatment with all the attendant costs is often the only effective option. Dental pus, it seems, is second-class pus.

"So why is it that for a preschool aged child, a boil on the bum is regarded so differently to a boil on the gum? ..."

Perhaps, because we know that oral diseases are largely preventable, there is a presumption of patient and parental culpability. I have heard my respected dental colleagues say… "If they'd only brushed their kid's teeth", "The bottle caused that", "They must have a rotten diet", "You should have come earlier", "You should be ashamed", "You didn't look after your child's teeth, why should the public purse pay to fix them?".

At a time when our countries are facing an epidemic of childhood obesity, children have signs of type 2 diabetes and early changes of heart disease, driven by many of the same dietary factors which promote caries, such thinking could dramatically cut our health budget! Imagine withdrawing public funding for treatment of preventible diseases!

Of course, such thinking ignores the knowledge that social disadvantage is at once both a risk factor for most diseases including Early Childhood Caries (ECC), and at the same time, a barrier to accessing treatment. There is a strong issue of social justice in the public support of population health, which cannot be ignored. Fortunately, New Zealand and Australia have been world leaders in the development of school based dental services and can be justifiably proud of their record in this area. However, despite the benefits of community fluoridation (all be it patchy), and after thirty years of steadily declining caries prevalence in children, there is now evidence that ECC is on the rise!

How should we as a profession, as educated leaders of the community, respond to this?

If the ever-benevolent tooth fairy were to wave her magic wand and give a huge bag of money to the health authorities, what would be the form of a dental service specifically targeted at ECC? Should it be an auxiliary based service, a dentist based service, a specialist based service, or a hybrid? Would it be best to develop a reparative service for preschoolers, or attempt to address the health beliefs of new parents at a time when they are receptive to new ideas and behavioural change? Perhaps we could just expand our acute care services for extractions. After all, they're only baby teeth!

The reality is that many of these kids will not be tractable patients, and many will need sedation or general anaesthesia to provide restorative treatment or extractions. Effective restorative management is likely to require full coverage, and would certainly require a high level of diagnostic and technical skill.

Perhaps the place to start would be antenatal classes, and then public maternity wards, to get oral health information to families before their children are infected with ECC. There is still much misinformation in the community about when (and indeed whether) preschoolers should have their first dental check-up. The concept of the 'well baby' check-up at twelve

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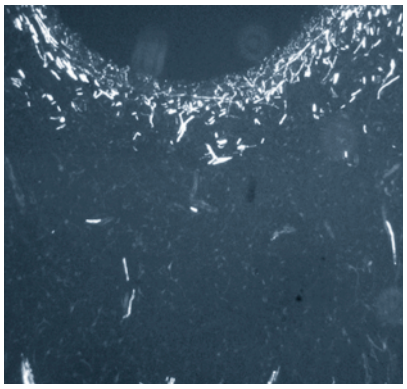
months of age is strongly supported by expert paediatric dental groups as an opportunity to prevent ECC, or at least to retrieve the situation with a remineralisation program rather than a restorative solution. Unfortunately, our adult focussed colleagues are often guilty of telling parents that their kids are too young to be seen. Perhaps they should come back when they are three, or perhaps even wait until they get to preschool. How many times have you heard parents tell of their attempts to get treatment, or even just an examination, for their child, only to be advised that they shouldn't worry about the teeth going chalky, or that things will be fine if they encourage their toddler to brush their (own) teeth! There is a vague awareness of the dangers of nursing bottles, but little attention to other forms of slow discharge feeders such as spill proof trainer cups and sport drink bottles, and even less attention to the alarming but increasingly prevalent inability of children to drink water!

Some of these issues would appear to overlap with the development of the many nutrition related medical problems facing our children, and perhaps we should look first to the broader issues of community health, rather than trying to break down the status quo to get universal public funding of reparative dental care. There is however a justifiable case for trying to improve oral health funding targeted at specific risk and special needs groups.

I look forward to seeing you all in Sydney in November for the IAPD Congress.

Dr John Winters

Taking advantage of some of the modern staining and image analysis techniques available, I decided to compare the overall innervation density of healthy and decayed primary and permanent teeth and compare their substance P expression, thus gaining some insight into their ability to process painful stimuli. I carried out a quantitative and qualitative analysis of the pulps of 120 extracted intact and carious primary and permanent molars. Essentially, there was no significant difference in the density of nerves in primary and permanent teeth, except in the mid-coronal region, where primary teeth had much smaller nerve trunks (Rodd and Boissonade, 2001), which probably just reflects the smaller pulp size of primary molars.



A. Labelling for the general neuronal marker PGP 9.5 in the pulp of a non-carious primary molar

B. Labelling for the general neuronal marker PGP 9.5 in the pulp of a carious primary molar showing increased neural density and branching between the pulp horns

A really interesting finding, however, was the fact that both primary and permanent teeth showed an intense increase in neural density with caries, as shown in the figures below. One may have expected that painful carious teeth would have a greater density of nerves than non-painful teeth, but this was not the case.

What I did find, however, was that the expression of substance P was greatly increased in painful samples as compared to non-painful carious samples (Rodd and Boissonade, 2000). An interesting finding was that there was little difference in substance P expression between primary and permanent teeth.

So to summarise these key findings:

- primary teeth have a similar overall innervation density to permanent teeth with the exception of having smaller nerve trunks
- both dentitions show marked increases in innervation density with caries progression
- Substance P is similar in both dentitions and is increased in painful carious samples

If we relate these findings to our clinical practice, there would seem to be no biological justification for not giving children local anaesthetic for cavity preparation in primary teeth (unless they are close to exfoliation, when profound neural degeneration will have occurred). Furthermore carious primary teeth seem to show similar defence and regeneration potential, in terms of their neural changes, to those seen in permanent teeth.

The second area of biological and clinical interest relates the pulp's inflammatory response to caries. A number of paediatric dentistry texts have suggested that primary pulps undergo a more pronounced and widespread inflammatory reaction than is seen in permanent teeth, and to some extent this has directed our clinical management of grossly carious primary teeth. However, there have been little biological data to substantiate this view.

So my second area of investigation was to compare two key components of

inflammation, vascularity and immune cell accumulation in primary and permanent teeth.

The key findings were that:

- primary teeth were more vascular than their successors in the mid-coronal region, but had a similar vascularity in other pulpal regions (Rodd and Boissonade, 2005)
- with caries progression, both dentitions showed a similar degree of vasodilatation and new vessel formation, which was predominantly in the pulp horn region
- primary teeth did contain a greater number of immune cells in both the intact and carious state. However, they appeared to localise immune cell accumulation, in the same way as permanent teeth (Rodd and Boissonade, in press).

I can only really speculate as to the clinical significance of these findings but I would not think that increased vascularity would be detrimental to the healing process in primary teeth. It's more likely to reflect the increased nutritional demands of the primary teeth and the wider apical foramina. Also, primary teeth showed good evidence of localising inflammatory changes and therefore the idea that they undergo widespread and uncontrolled pulpal inflammation seems unjustified.

Clinical management

From the findings discussed above, it would appear that primary and permanent teeth share a remarkably similar innervation, vascularity and inflammatory response to caries. So one would assume that their potential to heal after pulpal exposure or injury would also be similar. But our treatment approaches for the two dentitions are very different. With a carious exposure in a permanent tooth, every effort is made to preserve the vitality of the pulp, giving it the opportunity to regenerate and heal. In contrast, for the cariously exposed primary tooth pulp, we have tended to employ formocresol, thereby rendering the radicular pulp inert. I think it is time we asked ourselves whether the clinical procedures we use bare any relationship to our understanding of primary tooth pulp biology and pathophysiology? The range of clinical approaches for the management of the grossly carious vital primary tooth will now be briefly appraised.

Indirect pulp capping

The rationale behind indirect pulp capping is to maintain pulpal vitality in the hope that healing and repair will occur following gross caries removal. This approach has gained a recent increase in popularity with good long-term success rates (Al-Zayer *et al*., 2002; Vij *et al*., 2004). The aim is to carefully remove soft 'infected' dentine, whilst avoiding a pulpal exposure. It is usually recommended as a one-stage process. A calcium hydroxide or glass ionomer lining is then placed over the remaining stained dentine.

The success of this technique undoubtedly relies on appropriate case selection. It should only be undertaken on asymptomatic teeth with no clinical or radiographic signs of pulpal degeneration. Furthermore, it is absolutely critical that subsequent microleackage and bacterial invasion is avoided, otherwise pulpal inflammation and ultimately pulp necrosis will occur. This is achieved by placing a preformed crown or adhesive restoration.

Direct pulp capping

A direct pulp cap appears to have a very poor success rate in the primary dentition. It should only ever be considered for asymptomatic teeth where a minimal traumatic pulp exposure has occurred. It is then critical that the pulp is kept isolated from possible oral contamination prior to placement of a calcium hydroxide lining. However, internal resorption and necrosis are common reasons for failure. Interestingly, the use of mineral trioxide aggregate (MTA) as an alternative direct pulp-capping agent has recently been described (Bodem *et al*., 2004).

Vital pulpotomy

Here the aim is to remove the infected coronal pulpal tissue, leaving behind vital radicular tissue, which has the potential for healing and repair. Broadly speaking, there are three approaches: rendering the pulp tissue inert, preserving the tissue or actually encouraging tissue regeneration.

Over the years, formocresol has proved the most popular medicament for rendering the pulp inert. Formocresol contains formaldehyde thus fixes or denatures vital pulp tissue. It also has bactericidal properties so acts as a disinfectant. There is no doubt that the formocresol pulpotomy has achieved high levels of clinical success. However, the dental profession has always acknowledged that there are concerns about its' potential mutagenic, carcinogenic, and toxic effects.

In June 2004, a press release from the International Agency for Research on Cancer stated that there was now sufficient evidence that formaldehyde causes nasopharyngeal cancer in humans (www.iarc.fr). Following this, my Sheffield colleagues and I took the decision to remove formocresol from our clinic. This was admittedly a 'knee-jerk' reaction but we felt that, until further specific guidance was issued, we would stop using formocresol.

Interestingly a recent Brazilian study also highlighted the potential for formocresol to induce mutagenic changes (Zarzar *et al*., 2003). Blood samples were taken from 20 children before, and 24 hours following, a single formocresol pulpotomy. The number of chromosome abnormalities was then counted in cultured lymphocytes. Although there was no significant increase in chromosome abnormalities after the pulpotomy in the population as a whole, one child did show a six-fold increase in chromosome abnormalities, suggesting that, in his case, the formocresol had been mutagenic. Thus there may be individual susceptibly to the systematic effects of formocresol, even when used in small amounts.

The second vital pulpotomy approach aims to preserve radicular tissue by only inflicting a minimal inflammatory insult to the superficial radicular pulp. This is where ferric sulphate comes into play.

Ferric sulphate essentially acts as a haemostatic agent. When it is applied to the radicular pulp the iron combines with proteins in the blood to form a clot. This resultant 'barrier' then protects the underlying vital pulp. Success is similar to that achieved with formocresol (Fuks *et al*., 1997; Casas *et al*., 2004).

Finally, we move on to the 'ideal' type of pulpotomy, where the aim is to encourage tissue healing and dentine bridge formation at the site of pulpal amputation. Proposed medicaments include calcium hydroxide, mineral trioxide aggregate and bone morphogenic proteins.

In principle, calcium hydroxide should have provided good results but

unfortunately failure rates have proved much higher than with other treatments. Success rates at the worst have been 30% and at their best 80%. The main sequela has been internal resorption, which is believed to result from a failure to arrest bleeding at the amputation site.

One of the newest pulpotomy agents to emerge is MTA. This was first was approved in the States as a therapeutic endodontic material for humans in 1998. It comes in a grey or white formulation and has been used successfully in permanent teeth for direct pulp capping, apexification and treatment of lateral root perforations. It is biocompatible and actively stimulates hard tissue formation. Very recently, it was used in a prospective primary pulpotomy study where grey MTA achieved 100% success at six months (Agamy *et al.*, 2004).

For completeness, I will briefly mention bone morphogenic proteins, a generic term for a wide variety of bone signalling molecules. The most relevant to us in dentistry seems to be BMP7 (also known as osteogenic 1 protein). This has been cloned and produced from human DNA and has been used in animal models to achieve very good dentine bridge formation following pulp exposure (Six *et al.*, 2002). It therefore may have future applications for primary molar pulp therapies in humans.

Evidence-based practice

Faced with all these options, what works best? Unfortunately, as identified by a recent Cochrane Review, there are very few studies from which we can draw any reliable information (Nadin *et al.*, 2003). In fact this systematic review only identified three prospective randomised controlled trials that evaluated the success of primary pulpotomies over a twelve month period or more. From these studies, formocresol, ferric sulphate, electrosurgery pulpotomy and zinc oxide eugenol pulpectomy performed equally well. The review also highlighted the paucity of good clinical research in this area.

"...we should be looking towards pulp regeneration therapies particularly since it would appear that the primary tooth pulp has good potential for tissue repair and healing."

The current UK situation

Prior to the IARC press release, the vast majority of British Paediatric Dentistry Specialists favoured the use of formocresol (1:5 dilution) for the vital primary molar pulpotomy (Hunter and Hunter, 2003). However, current attitudes and practices are now very much divided. We are currently in the process of re-writing the British Society of Paediatric Dentistry Clinical Guidelines on primary molar pulp therapy, which will hopefully clarify the situation and identify best practice.

Conclusion

So what directions should we be taking with our primary pulp therapy? I personally believe that the era of empirical pulp treatment with non-biologically compatible medicaments or approaches has come to an end (no matter how good success rates have been). Instead, we should be looking towards pulp regeneration therapies particularly since it would appear that the primary tooth pulp has good potential for tissue repair and healing. The challenge remains for us to conduct rigorous laboratory and clinical-based research in order to help direct the best evidence-based practice.

Acknowledgements

Thanks are given to the Royal College of Surgeons of England and the Medical Research Council for research funding. I would also like to thank Nicky Kilpatrick, Sally Hibbert, and Sue Springbett for inviting me to speak at this meeting and to the AAPD for the great hospitality and welcome I received during my memorable trip to Australia.

References

Agamy HA, Bakry NS, Mounir MM, Avery DR (2004). Comparison of mineral trioxide aggregate and formocresol as pulp-capping agents in pulptomized primary teeth. *Pediatric Dentistry* 26: 302-309.

Al-Zayer MA, Straffon LH, Feigal RJ, Welch KB (2002). Indirect pulp treatment of primary posterior teeth: a retrospective study. *Pediatric Dentistry* 25: 29-36.

Bodem O, Blumenshine S, Zeh D, Koch MJ (2004). Direct pulp capping with mineral tioxide aggregate in a primary molar: a case report. *International Journal of Paediatric Dentistry* 14: 376-379.

Casas MJ, Kenny DJ, Johnston DH, Ludd PL (2004). Long-term outcomes of primary molar ferric sulfate pulpotomy and root canal treatment. *Pediatric Dentistry* 26: 44-48.

Fuks AB, Holan G, Davis JM, Eidelman E (1997). Ferric sulfate versus dilute formocresol in pulpotomized primary molars: a preliminary report. *Pediatric Dentistry* 19: 327-330.

Hunter ML, Hunter B (2003). Vital pulpotomy in the primary dentition: attitudes and practices of Specialists in Paediatric Dentistry practising in the United Kingdom. *International Journal of Paediatric Dentistry* 13: 246-250.

Nadin G, Glenny AM, Goel B, Yeung A (2003). Pulp treatment for extensive decay in primary teeth. The Cochrane Library. www.cochrane-oral.man.ac.uk

Rodd HD, Boissonade FM (2000). Substance P expression in human tooth pulp in relation to caries and pain experience. *European Journal of Oral Science* 108: 476-474.

Rodd HD, Boissonade FM (2001). Innervation density of human tooth pulp: a comparative study. *Journal of Dental Research* 80: 389-393.

Rodd HD, Boissonade FM (2005). Vascular status in human primary and permanent teeth in health and disease. *European Journal of Oral Sciences* 113: 1-7.

Rodd HD, Boissonade FM (in press). Caries-induced leucocyte responses in human primary and permanent teeth. *International Journal of Paediatric Dentistry*.

Six N, Lasfargues J, Goldberg M (2002). Differential repair responses in the coronal and radicular areas of the exposed rat molar pulp by recombinant human bone morphogenic protein 7. *Archives of Oral Biology* 47: 177-187.

Vij R, Coll JA, Shelton P, Farooq NS (2004). Caries control and other variables associated with success of primary molar vital pulp therapy. *Pediatric Dentistry* 26: 214-212.

Zarzar PA, Rosenblatt A, Takahashi CS, Takeuchi PL, Costa Junior LA (2003). Formocresol mutagenicity following primary tooth pulp therapy: an in vivo study. *Journal of Dentistry* 31: 479-485.

Dentinogenesis imperfecta: a review and a call for participation

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Introduction

Dentinogenesis Imperfecta (DI) is an inherited condition that affects the structure and appearance of dentine. In addition to the significant tooth wear, the teeth in affected individuals are discoloured and are aesthetically unsatisfactory. In addition, the treatment of these teeth is challenging, costly, time consuming and ultimately relatively unsuccessful. In many cases, the affected individuals will have most, if not all, their teeth removed before they reach adulthood.

The criteria by which a diagnosis of Dentinogenesis Imperfecta is arrived at are confusing and unclear. Whilst the presence of discoloured primary and permanent teeth appears to be uniform the occurrence of radiographic and other anomalies vary. Furthermore not only does the severity of tooth tissue loss, the spontaneous development of dental abscesses and the histological appearance of dentine vary but the relationship between DI and other comorbidities (such as sensorineural hearing loss) is unclear[1]. A mutation in chromosome 4 has been reported in some but not all affected families. Other possible mutations have yet to be identified. It is interesting that the level of severity of DI can be highly varied within a particular family, let alone between different families. It has been suggested that there is a genotype-phenotype correlation in DI however to date there has been no comprehensive study of the clinical, radiographic, histopathological and genetic data of broad spectrum of families presenting with DI Type II.

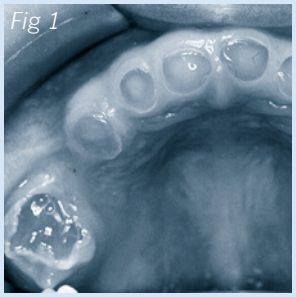
This short paper summarises the current literature on DI, identifies the gaps in knowledge and introduces a new and exciting project being carried out at the Murdoch Children's Research Institute in Melbourne and for which we are looking for assistance in identifying families who may be interested in participating.

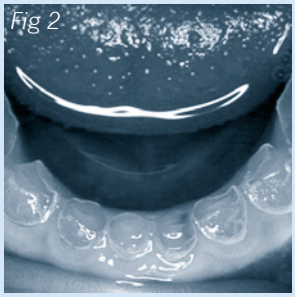
Classification of DI

In 1973, Shields proposed three types of dentinogenesis imperfecta[2]. Type I is the dentinal defect associated with osteogenesis imperfecta (OI). Type II, is the dentinal defect without the bone disease. Type III is a type of DI found exclusively in the 'Brandywine isolate' of Maryland. They are a triracial subpopulation consisting of Native American Indians, African Americans, and Caucasians of European decent. The findings of various studies suggest that DI II and DI III may clinical variations of the same genetic disease[3,4]. Although the need for revision of the classification of DI had been raised, Shield's classification is still currently used. In this literature review, only DI II will be discussed and so will be referred to as DI throughout this review.

Presentation of DI

The classic presentation of DI is the discoloration of teeth. The teeth demonstrate yellow/brown or opalescent grey discolouration. The discoloration stems from the abnormal composition of dentine, the abnormal dentine fails to support the overlying enamel. When enamel chips off from dentine, there will be severe wearing down of the exposed dentine upon oral function *(Figure 1 and 2)*. The yellow/brown form is more prevalent and more prone to attrition than the opalescent grey form[5,6]. The most striking radiographic features of DI are the bulbous crowns with constricted, short roots and progressive obliteration of the pulp chambers *(Figure 3)*. Periapical radio-lucent areas may also be noted in the absence of any other bone abnormality[7,8].





Figures 1 and 2: The severe wearing down of the exposed dentine in a 4-year-old with DI. Teeth 54 and 64 were extracted due to previous abscesses.

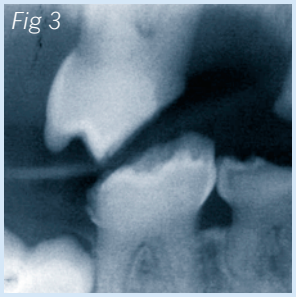


Figure 3: Same patient as Fig 1&2 with bulbous-shaped crowns, shortened roots and almost complete obliteration of pulp chambers and root canals of all primary teeth.

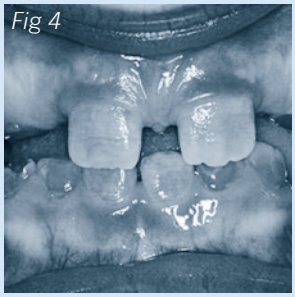


Figure 4: A 7-year-old DI with more severe wearing in primary teeth while the erupted permanent incisor and molars have not shown signs of attrition.

Primary teeth are more severely affected than the permanent dentition in DI[9-11]. Attrition in the primary teeth is rapid, leading to pulpal exposures as early as three years of age[12]. It is speculated that the DI gene is expressed more markedly in the primary teeth than the permanent ones[13] *(Figure 4)*.

Diagnosis of DI

The diagnosis of DI is worth discussing. Discoloration of teeth appears to be the minimum diagnostic criteria. Having radiographic presence of denticles, pulpal obliteration, and bulbous-shaped crowns, but absence of tooth discoloration, does not fulfill the diagnostic criteria. It appears that the radiographic features are overruled by the clinical ones[14]. However, it could be argued from a molecular genetics and biochemical perspective, that too discoloration is an inappropriate criterion on which to base a diagnosis[14]. To day there are no clear-cut criteria for the diagnosis of DI and it remains a clinical subjective judgment with no real consensus amongst clinicians concerning the diagnosis.

Linkage studies for DI gene

The search for the gene causing DI was a lengthy process which spanned 30 years. The first linkage study of DI was carried out in the late 1960s[15]. Despite several lengthy studies on large families no linkage to any recognised markers was reported[12]. In 1982, the group specific component (Gc), a vitamin D binding protein, was investigated more closely using a method of Gc subtyping[16]. Linkage of Gc with DI was reported, Gc being located on the long arm of human chromosome 4 (chromosome 4q).

Further linkage studies were carried out 10 years later by Crall[17]. Crall performed multipoint linkage analysis and conclusively established that the DNA marker INP-10 is tightly linked to DI. INP-10 is located in chromosome 4q21. Crall concluded that the genetic locus for DI must reside somewhere within the 10 million base pairs of DNA that surround the INP10 locus. Nevertheless, these studies provided only an approximate location for the DI gene.

The identification of DSPP gene

Gene mapping of DI was commenced in 1995. With the use of highly informative short tandem-repeat polymorphisms markers[18], the most likely position for the DI locus was identified as the 6.6 centiMorgans (cM) interval between markers D4S2691 (proximally) and D4S2692 (distally) refined to the chromosome 4 region 4q21-q23.

The DI disease region in chromosome was further refined in the study carried out by Aplin in 1999[19]. Subsequently, other dentally related genes were identified in the region which maps the critical region of DI[20-22]. However, none of these were shown to be the cause of DI.

Eventually, the discovery of two dentin-specific proteins called dentin sialoprotein (DSP) and dentin phosphoprotein (DPP), led to the identification of DSPP gene causing DI. The functions of these two proteins have been studied. Both DSP and DPP are believed to be essential for the formation of normal dentin[23]. DSP and DPP were later suggested to be the specific cleaved products of a single protein which is encoded by a single gene[24]. This gene was first named dentin sialophosphoprotein (DSPP) [24]. Since then, DSPP has become a strong candidate gene for DI II.

DSPP has been mapped using fluorescence in situ hybrdisation (FISH) and been assigned to chromosome 4 band q21.3[25]. Further refinement of the mapping was achieved by gene content mapping, as well as refining DSPP gene on the physical map of human chromosome 4 by sequence tagged site (STS) content mapping[8]. The fact that DSPP gene is located in the DI critical region of chromosome 4, and that it has an important function in formation of dentin, makes it a strong candidate gene for DI.

Mutations of DSPP gene

Since DSPP is a strong candidate gene for DI, it has become the choice for mutational analysis in DI families. By sequencing the DSPP, all the mutations identified in the Chinese families studied by Zhang[26] and Xiao[1] were contained within the DSP domain of the DSPP gene. These mutations have not been found in the Caucasian DI families of European decent that were studied by MacDougall (MacDougall's personal observation). MacDougall suggested the possible presence of other unidentified dentin/bone genes within the DI locus in chromosome 4 that may be contributory to DI. What was not suggested is that there may be other potential genes that may cause DI, lying on totally different chromosomes to chromosome 4.

DI and osteogenesis imperfecta

From the current classification of DI, there is a type of DI (DI type I, DI I), which is associated with osteogenesis imperfecta[2]. OI is also known as 'brittle bone disease' and it is a genetically inherited bone disorder. Although the clinical and radiological dental manifestations of DI I and DI type II (DI II) described throughout the literature are similar[27,28], their genotypic aetiology is completely different[18,29]. The majority of OI patients have mutations in the collagen 1 genes, COL 1A1 and COL1A2 on chromosomes 17 and 7 respectively[30]. About 10-50% of patients with OI have clinical signs of DI I[31-33] and indeed some believe that all OI patients have at least subclinical DI I[34]. These OI patients have mutations in the collagen 1 gene but no DSPP mutation and yet have the dental symptoms similar to individuals with DI II. Therefore, we speculate that patients with DI II who do not present with DSPP mutation, may have a collagen 1 gene mutation, giving the same dental presentation as DI I patients.

Conclusion

A review of the current literature surrounding Dentinogenesis Imperfecta confirms that several questions remain unanswered. There are obvious limitations in the current system of classifying DI. A system that is based upon an improved understanding of the molecular basis of DI would be helpful. Moreover, the current diagnostic criteria for DI are confusing and lack standardisation as they are based purely on subjective clinical and radiological examination. An improved understanding of the genetic basis of the disease will lead to more accurate diagnosis and classification. On a clinical level we are currently unable to provide families with information regarding the likely severity or potential life history of the disorder for their affected members nor on the impact it may have on future

Continued on page 10...

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Continued from page 7...

generations of their family. Improved understanding of the life history, the associated gene mutation(s) and the timing of their expression will help us, as clinicians, better inform our patients and ultimately provide better care.

The Research project

The aims of this project are:

1. To complete a comprehensive study of the phenotypic presentation of families with isolated dentinogenesis imperfecta
2. To screen DI patients for mutations in the DSPP gene.
3. To determine if DI patients who do not have DSPP mutations have mutations in the collagen I genes.

This study will facilitate the classification (and hence diagnosis) of DI based upon genetic aetiology rather than clinical manifestation. This has significant implications for both patients and their families as well as the healthcare professionals to whom they turn for management. Correlating the genotype with phenotype will enable clinicians to predict the severity of the disorder, provide information regarding its life history, determine the most appropriate treatment strategies and improve the health outcomes for this population.

Following approval from the Ethics in Human Research Committee at the Royal Children's Hospital in Melbourne, we have now started recruiting and examining families with isolated DI. We are keen to identify as many families in Australia as possible to participate in this project. We are seeking the help of our dental colleagues in identifying these families. Please contact Dr Kar Mun Chan if you feel you might be able to assist. She will discuss the practical issues involved including appropriate ethical consent, accessing the families etc.

Dr Chan can be contacted by

Telephone: 0414 288 787 or
(03) 9345 5462

Email: chankm98@yahoo.com

References

1. Xiao, S., *et al*., *Dentinogenesis imperfecta 1 with or without progressive hearing loss is associated with distinct mutations in DSPP.* [see comment][erratum appears in Nat Genet 2001 Mar;27(3):345]. Nature Genetics, 2001. 27(2): p. 201-4.
2. Shields, E.D., D. Bixler, and A.M. el-Kafrawy, *A proposed classification for heritable human dentine defects with a description of a new entity.* Archives of Oral Biology, 1973. 18(4): p. 543-53.
3. Kim, J.W., *et al*., *Mutational hot spot in the DSPP gene causing dentinogenesis imperfecta type II.* Hum Genet, 2005. 116(3): p. 186-91.
4. Boughman, J.A., *et al*., *An autosomal-dominant form of juvenile periodontitis: its localisation to chromosome 4 and linkage to dentinogenesis imperfecta and Gc.* Journal of Craniofacial Genetics & Developmental Biology, 1986. 6(4): p. 341-50.
5. Lund, A.M., *et al*., *Dental manifestations of osteogenesis imperfecta and abnormalities of collagen I metabolism.* Journal of Craniofacial Genetics & Developmental Biology, 1998. 18(1): p. 30-7.
6. O'Connell, A.C. and J.C. Marini, *Evaluation of oral problems in an osteogenesis imperfecta population.* Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, & Endodontics, 1999. 87(2): p. 189-96.
7. Gage, J.P., *et al*., *Hereditary opalescent dentine: variation in expression.* Journal of Dentistry for Children, 1991. 58(2): p. 134-9.
8. MacDougall, M., *Refined mapping of the human dentin sialophosphoprotein (DSPP) gene within the critical dentinogenesis imperfecta type II and dentin dysplasia type II loci*. European Journal of Oral Sciences, 1998. 106(Suppl 1): p. 227-33.
9. Luder, H.U., *et al*., *Mild dental findings associated with severe osteogenesis imperfecta due to a point mutation in the alpha 2(I) collagen gene demonstrate different expression of the genetic defect in bone and teeth.* Journal of Craniofacial Genetics & Developmental Biology, 1996. 16(3): p. 156-63.
10. Malmgren, B., M. Lundberg, and S. Lindskog, *Dentinogenesis imperfecta in a six-generation family. A clinical, radiographic and histologic comparison of two branches through three generations*. Swedish Dental Journal, 1988. 12(3): p. 73-84.
11. Mahoney, E.K., R.P. Widmer, and D.O. Sillence, *Opalescent dentine in two affected siblings.* New Zealand Dental Journal, 2001. 97(427): p. 15-8.
12. Mars, M., S. Farrant, and G.J. Roberts, *Dentinogenesis imperfecta. Report of a 5-generation family.* British Dental Journal, 1976. 140(6): p. 206-9.
13. Luder, H.U. and B. Steinmann, *Teeth in osteogenesis imperfecta. A mirror of genetic collagen defects?*, in *Studies in Stomatology and craniofacial biology.*, M.M.J. Cohen, B.J. Baum, and editors, Editors. 1997, IOS Press: Amsterdam. p. 209-228.
14. Kantaputra, P.N., *Dentinogenesis imperfecta-associated syndromes.[comment]*. American Journal of Medical Genetics, 2001. 104(1): p. 75-8.
15. Bixler, D., P.M. Conneally, and A.G. Christen, *Dentinogenesis imperfecta: genetic variations in a six-generation family.* Journal of Dental Research, 1969. 48(6): p. 1196-9.
16. Ball, S.P., *et al*., *Linkage between dentinogenesis imperfecta and Gc.* Annals of Human Genetics, 1982. 46(Pt 1): p. 35-40.
17. Crall, M.G., *et al*., *Genetic marker study of dentinogenesis imperfecta.* Proceedings of the Finnish Dental Society, *1992*. 88(Suppl 1): p. 285-93.
18. Crosby, A.H., *et al*., *Genetic mapping of the dentinogenesis imperfecta type II locus.* American Journal of Human Genetics, 1995a. 57(4): p. 832-9.
19. Aplin, H.M., K.L. Hirst, and M.J. Dixon, *Refinement of the dentinogenesis imperfecta type II locus to an interval of less than 2 centiMorgans at chromosome 4q21 and the creation of a yeast artificial chromosome contig of the critical region.* Journal of Dental Research, 1999. 78(6): p. 1270-6.
20. Crosby, A.H., *et al*., *Genomic organisation of the human osteopontin gene: exclusion of the locus from a causative role in the pathogenesis of dentinogenesis imperfecta type II.* Genomics, 1995b. 27(1): p. 155-60.
21. Crosby, A.H., *et al*., *Mapping of the human and mouse bone sialoprotein and osteopontin loci.* Mamm Genome, 1996. 7(2): p. 149-51.
22. MacDougall, M., T.T. Gu, and D. Simmons, *Dentin matrix protein-1, a candidate gene for dentinogenesis imperfecta.* Connective Tissue Research, *1996*. 35(1-4): p. 267-72.
23. Butler, W.T., *Dentin matrix proteins. European Journal of Oral Sciences, 1998.* 106(Suppl 1): p. 204-10.
24. MacDougall, M., *et al*., *Dentin phosphoprotein and dentin sialoprotein are cleavage products expressed from a single transcript coded by a gene on human chromosome 4. Dentin phosphoprotein DNA sequence determination.* Journal of Biological Chemistry, 1997. 272(2): p. 835-42.
25. MacDougall, M., *et al*., *Assignment of dentin sialophosphoprotein (DSPP) to the critical DGI2 locus on human chromosome 4 band q21.3 by in situ hybridisation.* Cytogenetics & Cell Genetics, 1997. 79(1-2): p. 121-2.
26. Zhang, X., *et al*., *DSPP mutation in dentinogenesis imperfecta Shields type II.[see comment]*. Nature Genetics, 2001. 27(2): p. 151-2.
27. Levin, L.S., *The dentition in the osteogenesis imperfecta syndromes.* Clinical Orthopaedics & Related Research, 1981(159): p. 64-74.
28. Shields, E.D., *A new classification of heritable human enamel defects and a discussion of dentin defects.* Birth Defects: Original Article Series, 1983. 19(1): p. 107-27.
29. Ranta, H., P.L. Lukinmaa, and J. Waltimo, *Heritable dentin defects: nosology, pathology, and treatment.* American Journal of Medical Genetics, 1993. 45(2): p. 193-200.
30. Byers, P.H., *Etiology of osteogenesis imperfecta: an overview of biochemical and molecular genetic analyses.* Connect Tissue Res, 1995. 31(4): p. 257-9.
31. Lukinmaa, P.L., *et al*., *Dental findings in osteogenesis imperfecta: I. Occurrence and expression of type I dentinogenesis imperfecta.* Journal of Craniofacial Genetics & Developmental Biology, 1987. 7(2): p. 115-25.
32. Sunderland, E.P. and C.J. Smith, *The teeth in osteogenesis and dentinogenesis imperfecta.* British Dental Journal, 1980. 149(10): p. 287-9.
33. Schwartz, S. and P. Tsipouras, *Oral findings in osteogenesis imperfecta.* Oral Surgery, Oral Medicine, Oral Pathology, 1984. 57(2): p. 161-7.
34. Hall, R.K., *et al*., *Odontoblast dysfunction in osteogenesis imperfecta: an LM, SEM, and ultrastructural study.* Connective Tissue Research, 2002. 43(2-3): p. 401-5.

Visit to the University of Papua New Guinea, Port Moresby

Dr Philippa Sawyer, Specialist Paediatric Dentist
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Clockwise from top left: 1. Fourth Year students receive oral hygiene instruction in a form which is most useful for child patients, Dr Perera is in the foreground. 2. A two year-old with recent trauma to the upper anterior teeth is examined in the knee-to-knee position. The patient's father is assisting. 3. A four year-old is having his first oral health check and students are learning to chart in FDI notation. 4. The fourth year students at the faculty.

On 7 August 2005 I travelled to Port Moresby to teach a week of paediatric dentistry to the fourth year students in the new Bachelor of Dental Surgery at the University of Papua New Guinea. I was met at the airport by Dr Perera and Dr Siddiqi who are two of the most dedicated academics with whom I have had the pleasure of spending time. The eight students in fourth year are treated as if they were their own children, as precious cargo. Together with Professor John McIntyre, they have struggled against all odds to put together the necessary equipment, materials, library resources and teaching staff. Two dedicated dental assistants work alongside the students and faculty to provide oral health care for the local population.

The students received lectures in the morning sessions and preclinical and clinical training in the afternoon sessions. They were shy at first, which is the local way, but warmed up very quickly displaying those wonderful smiles and gentle natures. The students were most attentive and displayed a great deal of interest in the behaviour management aspects of paediatric dentistry due to their previous experiences with children. They were fascinated by the pulpotomy and stainless steel crown exercises and eager to try their hands at crown preparations and crimping. The first three days were carefree with very comfortable teaching facilities, no power outages and reliable paediatric patients arriving for assessment with their parents.

Unfortunately the fourth day was marred by my succumbing to a dose of gastroenteritis from which I recovered quickly thanks to the kindness of all the staff and a local doctor, Dr Mark Paul, who trained at Royal Prince Alfred Hospital in Sydney. It was quite eventful and could happen to anyone, anywhere in the world. I am extremely grateful to everyone for their care and kindness. The final day was available for some questions and answers along with a class photo.

The experience was wonderful and I can recommend the trip to anyone who enjoys teaching highly motivated and enthusiastic students who are extremely receptive and appreciative of your efforts. The faculty is in search of an orthodontist who can provide undergraduate training in growth and development, diagnosis and treatment planning of malocclusion with an emphasis on removable appliance therapy. All enquiries should be directed to Professor John McIntyre.

john.mcintyre@adelaide.edu.au

ANZSPD Federal Secretary-Manager's report

1. ANZSPD has received correspondence from the Australian Dental Association Inc. regarding the requirement that, to remain affiliated with the ADA, all members of affiliated bodies must also be members of either the ADA or NZDA. This requirement has been called into question in recent times because it would appear to not be in accordance with Australian Competition and Consumer Commission [ACCC] guidelines. In 2003, the Australian Society of Orthodontists [ASO] successfully brought an application before the ACCC to enable this practice to be authorised. The ASO contended that the condition requiring ASO members to be ADA members was in the members' interest. They established that the services provided by the ADA to all its members, including ASO members, would not have to be duplicated by ASO in its service delivery to its members. Therefore, it was in the members' interest for the requirement of ADA membership to be retained.

The ADA sought preliminary legal advice which indicated the ACCC might approve similar applications by other affiliated bodies, and the ADA was offering the opportunity for these other societies [including ANZSPD] to be party to such an application. An approximate cost for this participation was quoted. It was decided for fairly obvious reasons that this was a matter which required a full and thorough discussion within our Society, and so it was agreed to not participate in the current action.

2. I have received comprehensive correspondence again from the ADA Inc. regarding the issuing of three new policy statements and the amending of four of their existing policy statements. Of these, one of the amended statements may be of interest to members – it is the Community Oral Health Promotion – Fluoride Use. The new policy statement: Delivery of Oral Health Care: Special Groups: Disabled Persons may also be of interest to members.

Alistair Devlin

ANZSPD – Branch News 2005

Western Australia

The first meeting of the year was held in May at the Australian Dental Association Inc. Lecture Theatre in West Perth. A short business meeting approved the new constitution, made necessary to satisfy the requirements to allow the branch to become incorporated. Incorporation has now happened. The presentation at the meeting was given by the newly appointed Senior Lecturer in Paediatric Dentistry at the University of Western Australia, Dr Boyen Huang. Boyen spoke on the topic of his Doctor of Philosophy thesis – 'Determinants of Traumatic Dental Injuries in Fifteen to Eighteen Year Old Students in Taiwan'. The thesis established an adolescent risk taking scale, which required those being studied to answer six questions with a simple three way choice: never, sometimes or often. It then looked at factors which influence the results, and three factors stood out. The first of these was family structure. If the adolescent lived with one or both birth parents, the chance of traumatic dental injury was less. Obviously, birth parents tended to be more protective; step parents were less likely to allocate financial resources to step children e.g. to provide trauma protection; and finally, because there was a distinct likelihood of there being higher psychosocial stress in a household with step parents and step children, greater risk taking behaviour occurred and a higher incidence of traumatic dental injuries followed.

The other two factors of significance were gender, with greater risk taking behaviour in males as expected; finally, socio-economic status gave the expected result, with those from lower socio-economic circumstances engaging in a greater degree of risk taking behaviour, with the resultant increase in traumatic dental injuries. Boyen looked at the nature of the injuries and also compared the incidence of dental injuries in Taiwan with other studies from around the world. In 2002, in Taiwan, 20% of adolescents had experienced some form of dental traumatic injury.

The branch has appointed an enthusiastic Local Organising Committee for the next ANZSPD. Federal Convention. This Committee has met a number of times and 'Congress West' has been appointed as the Professional Congress Organiser. The Cable Beach Club Resort in Broome, in the Kimberley region, has been booked for 23 – 27 May 2007. The Committee has been preparing for the launch of the promotion of the Convention at the IAPD Congress in Sydney in November.

Notice of General Meeting of the Society

In accordance with the Constitution of the Society, a General Meeting of ANZSPD has been called.

Date: 5.00pm Friday 4 November 2005

Venue: Sydney Convention and Exhibition Centre, Darling Harbour, Sydney

Business: 1. Presentation of Presidential and Financial Reports and 2. General Business

All members of the Society who will be in Sydney for the IAPD Congress are urged to attend.

Alistair Devlin

South Australia

We started off our program for the year with a day course, in conjunction with the Post Graduate Committee in Dentistry, as part of their recurrent education program. We aimed to help promote Paediatric Dentistry to the whole dental community, and attendees were from varied backgrounds, dentists in private practice, the public sector, dental therapists and several students in the dental course and Bachelor of Oral Health course.

The major speaker was Dr Nicky Kilpatrick, with two well received presentations on the Diagnosis and Management of Early Childhood Caries, and Dental Erosion. We thank Nicky for travelling to SA for us, and providing such good thought provoking material. The second presenter was a Cardiologist from the Women's and Childrens Hospital, Dr Malcolm Richardson. The third presenter was local endodontist Dr Ian Trantor, speaking on traumatic injuries. The day provided many opportunities to catch up with colleagues in very pleasant surroundings, and to have our knowledge updated.

Our next meeting was the usual evening format, with a guest speaker, Marg Pedler, to speak on Autism. Marg has recently set up a private centre for preschoolers to provide intensive behavioural and educational intervention to autistic children. It was an interesting evening.

Plans are underway for the RK Hall lecture next year in February or March, which we hope to hold in conjunction with our AGM.

Chris Adams

Queensland

The Annual General Meeting was held on Monday 7 February 2005. At the AGM the following officers were re-elected:

President	Robin Smith
Secretary/Treasurer	John Rutar
Federal representative	Kerrod Hallett
Committee Member	Michael Kenwood

Members thanked the Office Bearers for their tireless work during 2004.

The guest speaker at the AGM dinner meeting was Dr Steve Kazoulis, a final year MDSc student in Paediatric Dentistry. Steve spoke on the topic 'Dental Erosion in Young Children: Aetiology and Management'.

Further dinner meetings were held in April and July 2005. The guest speaker for the April meeting was Dr Vicki Law, final year MDSc student in Paediatric Dentistry, who spoke on 'Oral Colonisation of Children's Mouths with Mutans Streptococci'. Dr Steve Atkin, Specialist Orthodontist, Children's Oral Health Service, was the guest speaker for the July meeting. Steve spoke on the topic 'Evidence Based Practice in Dentistry' and approached the presentation in the same line as the current TV series – Mythbusters.

Future branch meetings are scheduled for September and November 2005. In September the dinner meeting presentation will be provided by Dr Liza Sansui and Dr Ola Al-Bataina who are final year MDSc students in Paediatric Dentistry. Liza and Ola will be speaking on the topic 'The Use of Lasers for Restorative Paediatric Dentistry'. The final dinner meeting for 2005 will be the 'President's at Home' which will be held at the United Service Club.

John Rutar

Victoria

At the ANZSPD Victorian Branch dinner meeting in May 2005, Dr Jocelyn Shand gave an interesting and well-illustrated presentation on recent advances in pediatric oral and maxillofacial surgery. Jocelyn is an Oral and Maxillofacial Surgeon with extensive experience in the treatment of children. She presented several case series of treatment in neonatal patients, whose care she had been involved in whilst working in the United States. In particular Jocelyn talked about the surgical treatment of severe airway obstruction and refractory sleep apnoea, showing graphic pictures of tongue lip adhesion and hyoid suspension, which have been used to treat these conditions. Jocelyn also illustrated osteogenesis distraction and resorbable fixation techniques, with specific reference to mandibular lengthening procedures, used in the treatment of neonates with micrognathia due to Robin Sequence and Nager Syndrome, and in children with craniosynostosis.

At the July dinner meeting Dr Andrew Kennedy from the Centre for Adolescent Health at the Royal Children's Hospital in Melbourne gave an enlightening talk entitled 'Why don't teenagers do what I ask? Getting them to follow medical advice'. He discussed the stages of psychosocial development that individuals go through as they progress through adolescence, and how this may be influenced by chronic illness and today's rapidly changing society. His talk highlighted the fact that knowledge and education are poor predictors of adherence to medical or dental advice, particularly in adolescents as they tend to be focused on the present rather than long term outcomes. Once patients and their families are reasonably well informed, further education is unlikely to change behavior. On a more optimistic note, Andrew suggested that adherence to medical or dental advice can be improved by simplifying treatment regimes, encouraging adolescents to be involved in decision making processes, ensuring clear expectations of treatment outcomes, and frequent monitoring. He reminded us that in order to foster a productive relationship between the dentist and the teenager trust, confidentiality and a non judgemental approach are essential.

At both evening meetings the dinner was preceded by a short presentation. In May this was given by Dental Health Services of Victoria, Health Promotions Division. They introduced 'Defenders of the tooth' – characters that are being used to promote the oral health message of eat well, drink well, clean well in the early childhood sector. In addition they provided information on the 'Donate a day' scheme, run between May and July 2005, in which dentists were encouraged to donate one hour of their time to talk to various groups on oral health issues. Dr Siew Luan Toh, a postgraduate student in pediatric dentistry, gave the presentation at the July meeting, highlighting various techniques used in vital bleaching.

Caroline Howarth

New Zealand

We have been having a quiet time, but working hard in our practices enjoying a milder winter.

Some of us have been involved in providing input for our local area health boards calling for comments on their 10 year strategic health plan. E.g. in Taranaki I have placed submissions to gain more early childhood interventions from our plunket nurses or maori health workers with part of their input being setting up good dental health programmes (not to put baby to sleep with a bottle, mothers to have breakfast as they set the pattern for the rest of the family, cleaning babies' teeth as soon as they erupt).

Nina Vasan, Peter Barwick (Orthodontist) and Mark Reddy (Paediatric Anaesthetist) gave a lecture at the Auckland Dental Association meeting in May on treating children under local, sedation and general anaesthetic. The meeting was well attended with over 140 dentists.

We are planning a lecture tour with Dr Tim Johnston (and a small part with Dr Nina Vasan) in June 2006. Proposed dates are Auckland, Friday 23rd; Christchurch, Saturday 24th; Dunedin Monday 26th; and Hamilton, Wednesday 28th. The contents will be aimed at therapists and general dentists as well as the association of dental therapists working in private practice. Topics covered are pulp therapy, use of lasers in paediatric dentistry and sedation. We look forward to having Tim relay his experiences in these areas and show him more of beautiful NZ.

I Look forward to catching up with everyone in Sydney soon!

MaryAnne Costelloe

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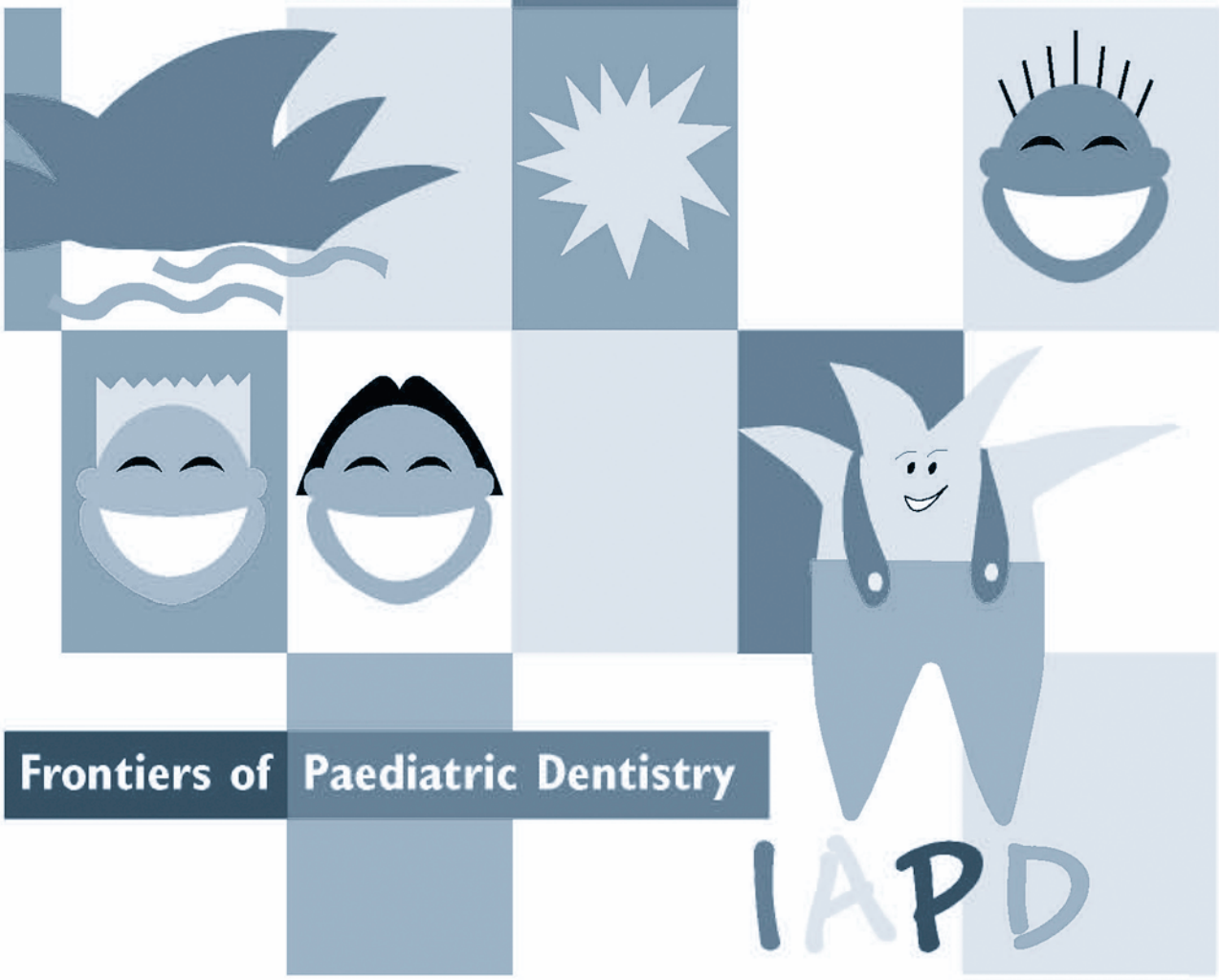

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IAPD 2005 Congress, 2–5 November, Darling Harbour

All eyes are turned towards the IAPD Congress which is just around the corner now! The organising committee and event planners are to be congratulated on the outstanding number of registrations for the conference, which far exceeds expectations. We look forward to seeing all of you at the conference! It will be an event to be remembered for many years. Don't miss out! And be sure to follow Dr Rabbit to the Colgate stand in the Exhibition to visit the friendly and ever helpful Colgate staff, to learn about Colgate products and the Colgate BSBF program, and to collect your free samples.

Colgate Bright Smiles Bright Futures

The Colgate *Bright Smiles Bright Futures* oral health education program will feature prominently on the Colgate stand at the IAPD Congress. If you do not already know about Colgate's long term commitment to oral health education through this innovative program, come to the Colgate stand at IAPD to find out. It has been a bumper year for the BSBF program in Australia/New Zealand and Dr Rabbit is very, very hoppy!

There has been an incredible response to the new Year 3 education materials – Dr Rabbit and the Legend of Tooth Kingdom – launched in February. More than 9,000 Year 3 kits were distributed to schools in Australia and New Zealand in response to the promotion. This means oral health education sessions were given to nearly 300,000 primary school children through the BSBF program this school year! At Colgate we are very proud of this accomplishment. Regrettably, the stocks of Year 3 kits for 2005 have now been exhausted, but kits will again be available from February 2006 for the 2006 school year!

As part of Colgate's Oral Health Month initiatives during August 2005, we ran a second *Bright Smiles Bright Futures* promotion. This one was targeted to all preschools encouraging oral health lessons with preschool students during August. More than 2000 Colgate *Bright Smiles Bright Futures* preschool educational kits were distributed in Australia and more than 1500 in New Zealand! That means more than 50,000 preschool children in Australia and more than 40,000 preschool children in New Zealand learned about good oral care during Colgate Oral Health Month.

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Coming events

31 October – 5 November 2005
20th IAPD International Congress
Sydney Convention and Exhibition Centre
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3-6 December 2005
1st International Workshop of the International Cleft Lip and Palate Foundation
Chennai (Madras), India

24-25 March 2006
Mediterranean Congress of Paediatric Dentistry
'Paediatric dentistry at 21st century; reality and prospects'
Palais des Congres, Marrakech, Morocco
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25-29 May 2006
59th AAPD Annual Session
Omni Hyatt and Westin Cincinnati, Ohio, USA

June 2006
5th PDAA Conference
Kaohsiung, Taiwan

23-27 May 2007
ANZSPD Federal Convention
Cable Beach Club Resort, Broome, WA

24-28 May 2007
60th AAPD Annual Session
Henry B. Gonzalez Convention Center
San Antonio, Texas, USA

14-17 June 2007
21st IAPD International Congress
Hong Kong Convention and Exhibition Centre
http://www.iapd2007.com/

8-13 June 2009
22nd IAPD International Congress
International Congress Centre, Munich, Germany

Stop press

Dr Katies Ayers has been awarded the title of *Outstanding Young Dentist of the Year* at the NZDA biennial conference in Napier this September. The reward recognises her academic and research achievements, including her contributions to the commisioned publications: *Development of a comprehensive public health approach to improving child oral health and reducing child health inequalities, 2004*, and *NZDA submission on Healthy Eating and fight the Obesity Epidemic*.

Austalian and New Zealand Society of Paediatric Dentistry

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Deadline next issue 20 January 2006